

# Latent classes of childhood trauma exposure predict the development of behavioral health outcomes in adolescence and young adulthood

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**Background.** To develop latent classes of exposure to traumatic experiences before the age of 13 years in an urban community sample and to use these latent classes to predict the development of negative behavioral outcomes in adolescence and young adulthood.

**Method.** A total of 1815 participants in an epidemiologically based, randomized field trial as children completed comprehensive psychiatric assessments as young adults. Reported experiences of nine traumatic experiences before age 13 years were used in a latent class analysis to create latent profiles of traumatic experiences. Latent classes were used to predict psychiatric outcomes at age  $\geq 13$  years, criminal convictions, physical health problems and traumatic experiences reported in young adulthood.

**Results.** Three latent classes of childhood traumatic experiences were supported by the data. One class (8% of sample), primarily female, was characterized by experiences of sexual assault and reported significantly higher rates of a range of psychiatric outcomes by young adulthood. Another class (8%), primarily male, was characterized by experiences of violence exposure and reported higher levels of antisocial personality disorder and post-traumatic stress. The final class (84%) reported low levels of childhood traumatic experiences. Parental psychopathology was related to membership in the sexual assault group.

**Conclusions.** Classes of childhood traumatic experiences predict specific psychiatric and behavioral outcomes in adolescence and young adulthood. The long-term adverse effects of childhood traumas are primarily concentrated in victims of sexual and non-sexual violence. Gender emerged as a key covariate in the classes of trauma exposure and outcomes.

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Exposure to childhood trauma is associated with a host of negative outcomes, including, but not limited to, post-traumatic stress disorder (PTSD). As demonstrated in investigations such as the Adverse Childhood Experiences (ACE) study (Felitti *et al.* 1998; Anda *et al.* 2006; Dube *et al.* 2009), childhood adversity has been associated with adult outcomes, such as suicide, depression, alcohol use, drug use, and physical consequences, such as autoimmune disorders. The

term ‘toxic stress’ has been used to describe multiple chronic stressors in childhood, including abuse, neglect, parental substance use, or parental depression, which may lead to changes in learning, behavior, and physiology that have an impact throughout adulthood (Garner & Shonkoff, 2012; Shonkoff & Garner, 2012). Childhood maltreatment has been suggested as a neurobiologically distinct subtype or ‘ecophenotype,’ due to the earlier onset of symptoms, symptom severity, co-morbidity, and poor treatment response associated with it (Teicher & Samson, 2013). Changes in reactivity in the hypothalamic–pituitary–adrenal (HPA) axis, reduced hippocampal volume and amygdala reactivity as well as epigenetic changes, such as DNA methylation, have been suggested as possible mechanisms for this relationship (Szyf, 2011; Teicher

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& Samson, 2013; Anacker *et al.* 2014). From a developmental perspective, diagnoses such as depression, PTSD and substance use disorder often have an onset in adolescence to early adulthood (Kessler *et al.* 2005). Childhood traumatic experiences may lead to changes in brain development, as well as gene expression, which influence the early development of behavior problems and onset of psychiatric symptoms. Thus, many mental and physical health diagnoses may have their roots in childhood experiences.

Due to the wealth of data on the negative impact of childhood traumatic experiences and the potential impact of stress on the developing brain, research in this area may benefit from isolating childhood trauma from trauma that occurs in adolescence or adulthood. It has been suggested that traumatic experiences in childhood lead to a more diverse array of PTSD symptoms than traumatic experiences that occur in adulthood (Cloitre *et al.* 2009). Epigenetic changes, such as DNA methylation, may depend on when in life the adverse experience occurs, as PTSD from a childhood trauma has a different epigenetic profile than PTSD from a trauma during adulthood (Mehta *et al.* 2013). Last, there is some evidence that childhood traumatic experiences early in life may increase the risk of trauma exposure later in adulthood (Acierno *et al.* 1999), the cumulative effect of which may have an impact on later outcomes in adulthood. Consequently, limiting the focus of analysis to the critical window of pre-adolescent experiences may help to tease apart potential influences on later development.

As defined by the DSM-5 diagnostic criteria of PTSD, trauma encompasses a range of experiences, from being directly assaulted to witnessing a traumatic event to hearing about a traumatic event experienced by a friend or family member (APA, 2013). Types of trauma exposures may have an impact on the development of negative outcomes, with experiences such as assaultive trauma conferring higher risk for PTSD and other psychiatric outcomes than other trauma types (Breslau *et al.* 1998a; Chung & Breslau, 2008; Wilcox *et al.* 2009). At the same time, the accumulation of multiple smaller traumatic and adverse experiences may also impact mental and physical health (Odgers & Jaffee, 2013). Exposure to one traumatic event in childhood may increase the likelihood of exposure to other childhood traumatic experiences, suggesting that childhood traumatic events are often interrelated (Dong *et al.* 2004). In addition, family history of psychopathology or substance use could create a familial environment in which multiple types of trauma are more likely to occur (Walsh *et al.* 2002, 2003). As a result, traumatic experiences may not occur in isolation and a pattern of multiple traumatic experiences across the same time period may be involved.

The current project aimed to study the effect of childhood trauma exposures before the age of 13 years on psychiatric (e.g. suicidal behavior, substance abuse), criminal, and physical health outcomes by young adulthood. A latent class analysis (LCA) approach permitted the examination of how different types of traumatic exposures typically cluster across childhood, in order to identify specific demographic and clinical profiles of childhood traumatic experiences. These classes can identify those individuals with childhood trauma who go on to develop negative outcomes in young adulthood, which will have implications for screening, intervention and prevention efforts. A wide breadth of negative outcomes in adolescence and young adulthood was investigated for a transdiagnostic perspective on the relationship of childhood trauma and psychiatric outcomes, as well as the potential impact of trauma on medical or behavioral outcomes. We used data from a community-based sample of children from an urban environment followed from childhood into young adulthood. Traumatic experiences were limited to those reported to occur before the age of 13 years in order to obtain a depiction of childhood adversity. Development of these latent classes can inform future genetic, epigenetic, and neurobiological studies of how specific types of childhood traumatic experiences lead to distinct negative outcomes in young adulthood.

## Method

### *Participants and procedures*

The sample consisted of 2311 participants initially recruited in first grade as part of an epidemiologically based, randomized field trial of two school-based preventive interventions, whose immediate targets were reading achievement and aggressive, disruptive behaviors (Kellam *et al.* 1991; Kellam *et al.* 2008). Nineteen elementary schools within five urban areas were randomly assigned within each urban area to a control condition or one of two intervention conditions provided only during first and second grade. The intervention conditions were the Good Behavior Game, which uses behavior management strategies to reduce impulsive, disruptive classroom behavior in a peer group context (Barrish *et al.* 1969) and Mastery Learning, a more precise, enhanced reading curriculum (Block & Burns, 1976). The 1815 participants that comprised the analytic sample completed at least one of four young adult assessments at approximately 19, 21, 22, and 29 years of age, and must have completed the list of childhood traumatic events used in the 1996 Detroit Area Survey (Breslau *et al.* 1998a), either at the 21- or 29-year assessment. Over half of

the sample was female ( $n = 957$ ; 52.7%), whereas 72% had a minority ethnicity (70.8% African American, 1.2% non-African American minority) and 55.2% received free or reduced meals in first grade. Participants included in the analytic sample did not differ from those not included in the initial sample by intervention status or age at entry into the study in first grade. However, the analytic sample was significantly more likely to receive subsidized lunch in first grade, to be African American and female (Storr *et al.* 2014).

This study was reviewed and approved by the Johns Hopkins School of Public Health Institutional Review Board. Written consent was obtained from parents for childhood participation for assessments beyond standard school assessments. Consent was obtained from each young adult at the time of each young adult interview. Participants completed surveys through interviews with research staff who were blind to previous assessments and received rigorous training prior to field work. Additional study details can be found in previous papers (see Storr *et al.* 2004; Wilcox & Anthony, 2004; Kellam *et al.* 2008; Wilcox *et al.* 2008).

## Measures

### *Child and young adult trauma exposure*

Participants completed the 1996 Detroit Area Survey on traumatic events to assess trauma exposure in childhood and young adulthood (Breslau *et al.* 1998b). Participants were asked if they had ever experienced any of 18 different traumatic events based on criteria A of the DSM-IV (APA, 2000). Items in this analysis are included in the Supplementary material.

Upon endorsing a traumatic event, participants responded to follow-up questions regarding their age at each event, which time was the worst experience, and age of worst experience. We defined any traumatic events occurring prior to age 13 as childhood traumas, whereas traumas occurring after age 18 represented young adult traumas, regardless of whether they were considered to be the 'worst experience'. Trauma exposure between ages 13 and 17 were not included in these analyses. Affirmative responses on these items in childhood and young adulthood during any assessment were combined to form the childhood and young adulthood trauma variables. Items with low base rates and similar content were combined to reduce model complexity (see caption of Fig. 1) into nine possible trauma exposure categories.

### *Parent psychopathology and substance use*

During the young adult assessments, participants were asked to recall characteristics of their primary

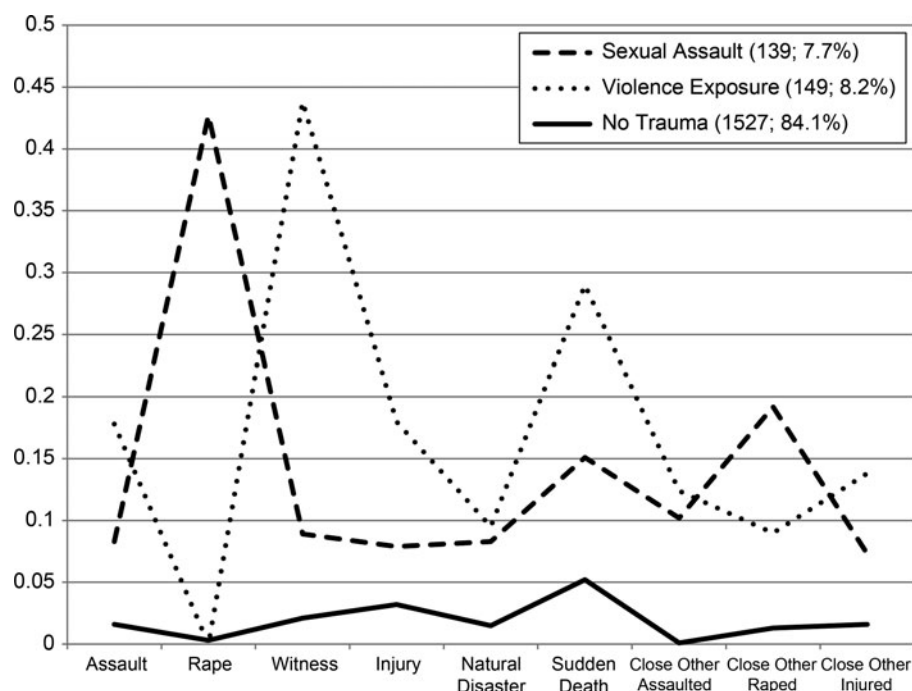
caregivers during their childhood, including parental substance use and psychiatric disorders. These items were modeled after similar items to assess family history in the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC; Grant *et al.* 2003). A 'yes' on any of four questions relating to parents (mother or father) having difficulties with drugs or alcohol was combined to form a dichotomous measure of parent substance use problems during childhood. Responses to three 'yes/no' questions about parents (mother or father) experiencing depression, mania, delusions or hallucinations, were combined to represent the presence of parental psychopathology during a participant's childhood.

### *Psychiatric and substance use disorder outcomes*

Participants were assessed for PTSD, major depressive disorder (MDD), antisocial personality disorder (ASPD), and alcohol and drug use disorders according to diagnostic criteria stipulated in the DSM-IV, text revision (APA, 2000). All waves used a Composite International Diagnostic Interview (CIDI)-based instrument which was fully structured to allow administration by lay interviewers and scoring of diagnoses by computer. The CIDI-UM (Kessler *et al.* 1994) was used in the age 19 assessment and subsequent waves used the World Mental Health Composite International Diagnostic Interview, Version 2.1 (WHO WMH-CIDI) (Kessler *et al.* 1994; WHO, 1997), following procedures from the National Comorbidity Survey (Anthony, 1994). The CIDI has demonstrated acceptable validity when compared to independent clinical interview (Wittchen, 1994; Andrews & Peters, 1998), as well as the PTSD module (Breslau *et al.* 1998b). Only those who met criteria for the diagnosis with onset at age  $\geq 13$  years during any of these assessments were indicated as having these diagnoses. Those who reported meeting criteria for the psychiatric diagnoses before the age 13 years were coded as missing data for these variables and were thus not included in evaluation of the outcomes. This procedure was used to maintain evaluation of the time ordered relationship between trauma exposure and psychopathology.

### *Suicide ideation and attempt*

Questions regarding suicide attempt or ideation were asked as part of the MDD module in all of the assessment waves (additional detail in Supplementary material). In order to constrain temporality, only those who endorsed attempt or ideation at age  $\geq 13$  years were coded as having had suicidal behavior. To account for suicidal behavior being a diagnostic criteria for MDD, a diagnosis of MDD at age  $\geq 13$  years was



**Fig. 1.** Item response probabilities for latent classes of trauma before the age of 13 years. Indicators for the classes were comprised of bundles of items given low response rates on multiple single items. Indicators included the following items: Assault – includes being captured/kidnapped, shot, stabbed, mugged or beaten up; Rape – includes rape or sexual assault; Witness – includes witnessing a death or discovering a dead body; Injury/Illness – includes motor vehicle accident, life-threatening illness, or other injury to self; Natural Disaster – includes experiencing a natural disaster; Sudden Death – includes experiencing the sudden death of a friend or family member; Close Other Assaulted – includes the physical attack of a family member or friend; Close Other Raped – includes rape or sexual assault of a family member or friend; Close Other Injured – includes motor vehicle accident, life-threatening illness or injury of a friend or family member.

operationalized without including the criteria regarding suicidal behavior.

#### *Criminal convictions*

Adult criminal records for participants were procured in 2004 from the adult prison database of Maryland Department of Corrections, when participants were 24–25 years old. Thus, these data reflect criminal convictions that occurred between the ages of 18–25 years for participants (additional detail in the Supplementary material). These data were stratified to represent violent, non-violent, and substance-related criminal convictions.

#### *Health outcomes*

The National Health and Nutrition Examination Survey III: Data Collection Forms (NHANES; National Center for Health Statistics, 1994) interviews have been developed for use in the ongoing National Center for Health Statistics studies. At the 29-year assessment, participants responded to items derived from the NHANES interview, which included ‘yes’/‘no’ screening items related to sexually transmitted

infections (STIs), diabetes, stomach ache, headache, and hypertension. During this assessment, height and weight were measured by trained staff with a scale and tape measure, from which body mass index (BMI) was estimated (i.e. weight (lb)/[(height (in)<sup>2</sup>) × 703]). BMI was categorized above and below 30, the cut point for obesity (WHO, 1995).

#### *Analysis*

A LCA was conducted using Mplus version 7.11 (Muthén & Muthén, 2013) to model childhood trauma experiences prior to age 13. An LCA is a person-oriented approach to identify distinct subgroups of individuals across a series of indicator variables. Analyses assumed that distinct groups exist based on the patterns of trauma exposures. Model fit and class viability were identified using comparison of goodness-of-fit indices and the distinctiveness of classes. Goodness-of-fit indices included the Bayesian Information Criterion (BIC), Vuong–Lo–Mendell–Rubin likelihood ratio test (VLMR-LRT; Lo *et al.* 2001) and bootstrap likelihood ratio test (BLRT; McLachlan & Peel, 2000). The auxiliary facility in

**Table 1.** Class enumeration for latent class analyses

No. of classes	df	LL	BIC	LRT		BLRT		Entropy	Smallest class <i>n</i> (%)
				$\Delta 2 \times LL$	<i>p</i>	$\Delta 2 \times LL$	<i>p</i>		
2	25	-3091.87	6371.38	212.21	0.007	213.98	<0.001	0.57	240
3	41	-3034.62	6376.97	113.55	0.005	114.50	<0.001	0.72	120
4	57	-3015.77	6459.36	37.39	0.86	37.40	0.33	0.55	113

BIC, Bayesian Information Criterion; BLRT, bootstrapped likelihood-ratio test; df, degrees of freedom; LL, log likelihood; LRT, Lo-Mendell-Rubin likelihood ratio test.

Mplus was used to evaluate class differences according to overall and pairwise differences in the outcome across classes with posterior probabilities from multiple imputations (Muthén & Muthén, 2013). The young adult outcomes included suicidal behavior, psychiatric diagnoses, and health outcomes occurring at age  $\geq 13$  years, as well as criminal convictions and traumatic events during young adulthood. Due to the relationship between trauma and PTSD, PTSD symptom clusters were also included as potential outcomes. For more information on LCA, please refer to Collins & Lanza (2009).

#### Covariates

Variables serving as covariates related to contextual, environmental characteristics and demographics and were included in the LCA models to identify the influence of concurrent childhood adversity experiences on trauma exposure. Contextual characteristics included intervention status (0 = control condition, 1 = Good Behavior Game or Mastery Learning), parental psychopathology, and parental substance use. Demographic controls included gender (0 = male, 1 = female), race/ethnicity (0 = European-American, 1 = African-American), and subsidized lunch status (0 = no, 1 = yes). Free and reduced meals status served as a proxy variable for low income.

#### Treatment of missing data

Missing data was managed with full information maximum likelihood (FIML) estimation. FIML produces parameter estimates that are less biased than other missing data strategies even when the data are not missing at random (Graham, 2009).

## Results

### Class enumeration

An LCA was estimated to identify typical patterns of trauma exposure during childhood in order to create

latent classes. Results from comparing models indicated that three classes provided the best fit for the data (see Table 1). Although the BIC increased as additional classes were extracted, other fit indices supported the three class model. Three classes were the highest number of classes for which the LRT and BLRT remained significant. Moreover, the entropy was highest for the three class model. The posterior probabilities of class membership demonstrated adequate class separation (range 0.029–0.180) and homogeneity of class (range 0.770–0.994).

The three classes corresponded to distinct and interpretable classes of trauma exposure before the age of 13 (see Fig. 1). The first class, the Low Childhood Trauma class ( $n = 1549$ , 85.2%), had low probabilities of trauma exposure. The second, Violence Exposure class ( $n = 149$ , 8.2%), displayed high rates of experiencing physical assault and physical injury, or witnessing physical assault, physical injury or death. The third, Sexual Assault class ( $n = 120$ , 6.6%), demonstrated the highest rates of rape and sexual assault, as well as having close friends or family also experiencing sexual assault.

#### Effect of control variables

A strong gender effect emerged, where men were significantly more likely to belong to the Violence Exposure class, and women were more likely to be members of the Sexual Assault class (see Table 2). Only young adults belonging to the Sexual Assault classes were significantly more likely to have had a caregiver with psychopathology during childhood ( $p < 0.01$ ) than the Low Childhood Trauma class. No other control variables were significantly different among classes.

#### Effect of trauma class membership on young adult outcomes

The childhood trauma classes displayed many differences across young adult outcomes (see Table 3). The

**Table 2.** Covariates of latent class membership

	Violence Exposure ( <i>n</i> = 149)	Sexual Assault ( <i>n</i> = 120)	Low Childhood Trauma ( <i>n</i> = 1549)	Violence Exposure <i>v.</i> Low Childhood Trauma		Sexual Assault <i>v.</i> Low Childhood Trauma		Sexual Assault <i>v.</i> Violence Exposure	
				<i>b</i>	<i>p</i>	<i>b</i>	<i>p</i>	<i>b</i>	<i>p</i>
<b>Demographics</b>									
Male gender	134 (90%)	16 (13%)	709 (46%)	<b>1.95</b>	<b>0.021*</b>	<b>-1.43</b>	<b>&lt;0.001*</b>	<b>-3.38</b>	<b>&lt;0.001*</b>
African American	110 (74%)	80 (67%)	1096 (71%)	0.25	0.46	-0.19	0.56	-0.44	0.37
Received subsidized lunch	81 (54%)	66 (55%)	857 (55%)	-0.29	0.27	0.08	0.77	0.37	0.33
<b>Contextual characteristics</b>									
GBG/ML intervention	56 (38%)	50 (42%)	666 (43%)	-0.16	0.52	-0.07	0.77	0.01	0.78
Parental psychopathology	40 (27%)	50 (42%)	329 (21%)	0.46	0.16	<b>0.66</b>	<b>0.008*</b>	0.10	0.64
Parental problems with drugs or alcohol	47 (32%)	52 (43%)	415 (27%)	0.28	0.35	0.30	0.22	0.03	0.95

GBG/ML, Good Behavior Game/Mastery Learning intervention; statistically significant parameter estimates are given in bold.

\*  $p < 0.05$ ; statistically significant parameter estimates are notated in bold text.

Sexual Assault class, but not the Violence Exposure class, had significantly higher rates of both suicide attempt, having an attempt plan, and ideation than the Low Childhood Trauma class. Those in both the Violence Exposure and Sexual Assault classes were significantly more likely to have a PTSD diagnosis than the Low Childhood Trauma class. By diagnostic symptom clusters of PTSD, only the Sexual Assault class demonstrated significantly higher likelihood of re-experiencing and avoidance symptoms than the Low Childhood Trauma class. The Sexual Assault class also had higher rates of MDD than the other two classes, whereas the Violence Exposure class had higher rates of ASPD as well as alcohol and drug use disorders than the other two classes. The Violence Exposure class was significantly more likely than the other two classes to have convictions for violent, non-violent and substance-related crimes. The Sexual Assault class was significantly less likely to experience substance-related crimes compared to both classes.

Childhood trauma classes were strong predictors of young adult trauma experiences. The Sexual Assault class had significantly higher odds of experiencing sexual assault during young adulthood than the other classes. Similarly, the Violence Exposure class had significantly higher odds of being physically assaulted during young adulthood than the other two classes.

The Violence Exposure class also had a higher likelihood of witnessing death than the Low Childhood Trauma class. Finally, only one of the physical health outcomes was significantly different among the classes. The Sexual Assault class was significantly more likely to disclose having had a STI than the other two classes.

#### *Gender effect of outcomes*

Given the strong effect of gender on child trauma class membership, we conducted follow-up analyses on gender differences in young adult outcomes. We conducted gender-specific  $\chi^2$  tests comparing the Violence Exposure class to the Low Childhood Trauma class among men and among women compared the Sexual Assault class to the Low Childhood Trauma class. We found marked differences in outcomes (see Table 4). The women in the sexual trauma class were significantly more likely to have experienced suicide attempt and ideation, all psychiatric diagnoses, stomach problems, and to have had a STI than women in the Low Childhood Trauma class. Men in the Violence Exposure class were more likely to experience ASPD and PTSD and to have had a non-violent criminal conviction, but did not show significant differences in other psychiatric outcomes

**Table 3.** Outcomes of latent class membership of child trauma experiences

	Violence Exposure	Sexual Assault	Low Childhood Trauma	Violence Exposure v. Low Childhood Trauma		Sexual Assault v. Low Childhood Trauma		Violence Exposure v. Sexual Assault	
				$\chi^2$	<i>p</i>	$\chi^2$	<i>p</i>	$\chi^2$	<i>p</i>
<b>Suicide outcomes</b>									
Attempts	12 (8%)	32 (27%)	119 (8%)	0.34	0.559	<b>14.03</b>	<b>&lt;0.001*</b>	<b>-7.18</b>	<b>0.007*</b>
Plan	9 (7%)	20 (17%)	88 (6%)	0.275	0.335	<b>13.06</b>	<b>&lt;0.001*</b>	<b>-7.25</b>	<b>0.006*</b>
Ideation	26 (17%)	41 (34%)	245 (16%)	1.07	0.302	<b>13.05</b>	<b>&lt;0.001*</b>	<b>-3.84</b>	<b>0.05*</b>
<b>Psychiatric diagnostic outcomes</b>									
ASPD	79 (53%)	49 (41%)	497 (32%)	<b>13.80</b>	<b>&lt;0.001*</b>	1.09	0.30	<b>4.60</b>	<b>0.032*</b>
MDD	13 (9%)	26 (22%)	195 (13%)	0.36	0.55	<b>5.35</b>	<b>0.02*</b>	<b>-5.68</b>	<b>0.017*</b>
PTSD	31 (21%)	38 (32%)	117 (8%)	<b>10.35</b>	<b>0.001*</b>	<b>22.53</b>	<b>&lt;0.001*</b>	-2.03	0.155
Re-experiencing	44 (30%)	43 (36%)	355 (23%)	2.33	0.127	<b>4.61</b>	<b>0.032*</b>	-0.14	0.709
Avoidance	9 (6%)	21 (18%)	79 (5%)	0.17	0.682	<b>6.92</b>	<b>0.009*</b>	-3.37	0.067
Hyper-arousal	16 (11%)	23 (19%)	158 (10%)	0.12	0.731	2.79	0.095	-1.02	0.313
Alcohol use disorder	59 (40%)	36 (30%)	433 (28%)	<b>8.29</b>	<b>0.004*</b>	0.04	0.85	<b>4.10</b>	<b>0.04*</b>
Drug use disorder	52 (35%)	32 (27%)	323 (21%)	<b>9.04</b>	<b>0.003*</b>	0.34	0.56	<b>4.24</b>	<b>0.04*</b>
<b>Physical health outcomes</b>									
BMI	mean = 28.08 s.d. = 5.75	mean = 30.55 s.d. = 8.04	mean = 30.14 s.d. = 7.35	-3.46	0.063	0.26	0.612	-3.08	0.079
Obesity (BMI ≥ 30)	33 (22%)	48 (35%)	454 (30%)	-0.81	0.37	1.25	0.263	-2.35	0.125
Diabetes	1 (1%)	7 (5%)	45 (3%)	-1.27	0.261	0.55	0.458	-1.77	0.184
Stomach problems	9 (6%)	18 (13%)	93 (6%)	0.07	0.80	3.50	0.061	-1.63	0.202
Severe headaches	32 (22%)	46 (33%)	334 (22%)	0.01	0.914	2.90	0.089	-1.41	0.235
Hypertension	12 (8%)	22 (16%)	159 (10%)	-0.01	0.945	1.34	0.248	-0.90	0.344
STI	35 (25%)	59 (44%)	363 (25%)	0.07	0.796	<b>10.00</b>	<b>0.002*</b>	<b>-6.86</b>	<b>0.009*</b>
<b>Criminal convictions</b>									
Violent crimes	65 (44%)	26 (22%)	388 (25%)	0.54	0.462	-0.35	0.557	0.97	0.326
Non-violent crimes	7 (5%)	11 (10%)	108 (7%)	0.38	0.537	-3.64	0.056	2.74	0.098
Substance-related crimes	67 (45%)	13 (11%)	354 (23%)	1.20	0.274	<b>-8.40</b>	<b>0.004*</b>	<b>9.97</b>	<b>0.002*</b>
<b>Young adulthood trauma (≥ 18 yr)</b>									
Sexual assault	3 (2%)	12 (10%)	33 (2%)	0.152	0.696	<b>6.55</b>	<b>0.010*</b>	<b>-5.82</b>	<b>0.016*</b>
Physical assault	52 (35%)	28 (23%)	355 (23%)	<b>5.74</b>	<b>0.017*</b>	0.23	0.629	<b>4.85</b>	<b>0.028*</b>
Witnessing death	54 (36%)	32 (27%)	295 (19%)	<b>9.94</b>	<b>0.002*</b>	0.99	0.319	2.90	0.09

ASPD, Antisocial personality disorder; MDD, major depressive disorder; PTSD, post-traumatic stress disorder; BMI, body mass index; STI, sexually transmitted infection.

Suicidal behavior and psychiatric diagnoses reflect onset from the age of ≥ 13 years; criminal convictions and young adult trauma reflect experiences from the age of ≥ 18 years.

\*  $p < 0.05$ ; statistically significant parameter estimates are given in bold.

including suicidal behavior, health outcomes, or other criminal convictions from men in the Low Childhood Trauma class. Men in the Violence Exposure class were more likely than men in the Low Childhood Trauma class to have witnessed another person's death and women in the Sexual Assault class were more likely than women in the Low Childhood Trauma class to experience all young adult traumatic events.

There were too few women in the Violence Exposure class and men in the Sexual Assault class to report

any additional analyses beyond descriptive results (Additional detail in the Supplementary material).

## Discussion

Three latent classes of childhood traumatic experiences before the age of 13 years emerged from an analysis of a community-based sample from an urban environment. The first group, which accounted for the majority (84%) of the sample reported low levels of all

**Table 4.** Gender effects of outcomes of latent classes of child trauma experiences

	Males only			Females only		
	Violence Exposure ( <i>n</i> = 134)	Low Childhood Trauma ( <i>n</i> = 709)	<i>p</i>	Sexual Assault ( <i>n</i> = 104)	Low Childhood Trauma ( <i>n</i> = 840)	<i>p</i>
<b>Suicide outcomes</b>						
Attempts	10 (7%)	40 (6%)	0.258	<b>28 (27%)</b>	<b>74 (9%)</b>	<b>&lt;0.001*</b>
Plan	8 (7%)	37 (6%)	0.439	<b>17 (16%)</b>	<b>51 (6%)</b>	<b>&lt;0.001*</b>
Ideation	22 (16%)	109 (15%)	0.414	<b>36 (35%)</b>	<b>136 (16%)</b>	<b>&lt;0.001*</b>
<b>Psychiatric diagnoses</b>						
ASPD	<b>76 (56%)</b>	<b>312 (44%)</b>	<b>0.005*</b>	<b>39 (36%)</b>	<b>185 (22%)</b>	<b>0.001*</b>
MDD	12 (9%)	58 (8%)	0.416	<b>27 (22%)</b>	<b>132 (16%)</b>	<b>0.033*</b>
PTSD	<b>26 (19%)</b>	<b>57 (8%)</b>	<b>&lt;0.001*</b>	<b>33 (31%)</b>	<b>60 (7%)</b>	<b>&lt;0.001*</b>
Re-experiencing	<b>40 (30%)</b>	<b>134 (27%)</b>	<b>0.010*</b>	<b>39 (43%)</b>	<b>221 (32%)</b>	<b>0.019*</b>
Avoidance	9 (7%)	31 (6%)	0.227	<b>20 (22%)</b>	<b>48 (7%)</b>	<b>&lt;0.001*</b>
Hyper-arousal	16 (12%)	56 (11%)	0.142	<b>22 (25%)</b>	<b>102 (15%)</b>	<b>0.015*</b>
Alcohol use disorder	57 (47%)	269 (41%)	0.142	<b>29 (28%)</b>	<b>164 (21%)</b>	<b>0.035*</b>
Drug use disorder	49 (41%)	218 (32%)	0.075	<b>25 (21%)</b>	<b>105 (13%)</b>	<b>0.002*</b>
<b>Health outcomes</b>						
BMI	mean = 27.93 s.d. = 5.37	mean = 29.19 s.d. = 6.15	0.051	mean = 30.36 s.d. = 7.97	mean = 30.80 s.d. = 8.01	0.638
Obesity (BMI ≥30)	23 (31%)	122 (35%)	0.347	34 (49%)	235 (46%)	0.331
Diabetes	1(1%)	9 (2%)	0.465	5 (6%)	37 (5%)	0.537
Stomach problems	8 (6%)	35 (7%)	0.472	<b>15 (16%)</b>	<b>60 (8%)</b>	<b>0.017*</b>
Severe headaches	28 (20%)	101 (20%)	0.083	34 (38%)	239 (33%)	0.294
Hypertension	11 (9%)	68 (14%)	0.269	17 (19%)	93 (14%)	0.106
STI	31 (23%)	137 (19%)	0.204	<b>46 (43%)</b>	<b>233 (27%)</b>	<b>0.001*</b>
<b>Criminal convictions</b>						
Violent crimes	61 (46%)	286 (40%)	0.556	19 (63%)	102 (62%)	0.539
Non-violent crimes	<b>7 (46%)</b>	<b>63 (42%)</b>	<b>0.045*</b>	8 (26%)	45 (27%)	0.563
Substance-related crimes	62 (46%)	288 (41%)	0.501	8 (26%)	66 (40%)	0.113
<b>Traumatic events in young adulthood</b>						
Sexual assault	1(1%)	1(1%)	0.293	<b>11 (9%)</b>	<b>32 (4%)</b>	<b>0.005*</b>
Physical assault	51 (38%)	246 (34%)	0.257	<b>24 (22%)</b>	<b>109 (13%)</b>	<b>0.006*</b>
Witnessing death	<b>52 (39%)</b>	<b>180 (25%)</b>	<b>0.001*</b>	<b>25 (23%)</b>	<b>115 (14%)</b>	<b>0.008*</b>

ASPD, Antisocial personality disorder; MDD, major depressive disorder; PTSD, post-traumatic stress disorder; BMI, body mass index; STI, sexually transmitted infection.

Suicidal behavior and psychiatric diagnoses reflect onset from the age of ≥13 years; criminal convictions and young adult trauma reflect experiences from the age of ≥18 years.

\*  $p < 0.05$ ; statistically significant parameter estimates are given in bold.

traumatic experiences. The second class comprised 8% of the sample, was predominately male, reported the highest levels of witnessing violence, physical assault, physical injury and the sudden death of a close friend or family member. The third group comprised 8% of the sample, which was predominately female, reported the highest levels of sexual assault for themselves and knowing someone who had been sexually assaulted. The second and third classes had different profiles of psychiatric diagnoses occurring at age ≥13 years as well as the same types of trauma exposures in young adulthood. Distinct patterns of outcomes emerged

when analyses were stratified by gender; women in the Sexual Assault group reported a wide range of negative psychiatric outcomes, while men in the violence exposure class reported a pattern of outcomes limited to post-traumatic stress and antisocial personality.

The creation of these latent classes suggests clustering of types of traumatic experiences (physical *v.* sexual assault) by gender. Differences in exposure to traumatic experiences by gender has been reflected in the Youth Risk Behavior Survey (YRBS), in which 10% of female and 4% of male high-school students



report that they have been forced to have sexual intercourse (CDC, 2015), as well as the ACE study in which 25% of the women and 16% of the men reported sexual abuse in childhood (CDC, 2014). Similarly, an analysis of the Detroit Area Survey of Trauma found that lifetime exposure to assaultive trauma and witnessing a death were more likely in men than women (43% *v.* 32% for assaultive violence; 40% *v.* 19% for witnessing death or violence) (Breslau *et al.* 1998a). This analysis further extends this literature to suggest that these gender differences in trauma exposure begin before the age of 13 years and reinforces the importance of gender as a critical construct in trauma research.

The relationship of parental psychopathology to the Sexual Assault class highlights the significance of familial environment and possible genetic effects on the association between childhood trauma and psychiatric diagnosis. On one hand, there is a robust literature linking parental psychopathology with abuse exposure in offspring (Chaffin *et al.* 1996; Walsh *et al.* 2002) with some analyses proposing that childhood abuse may, in part, mediate the intergenerational transmission between parent and child diagnoses (Verona & Sachs-Ericsson, 2005). On the other hand, recent evaluations have found interactions between genetic polymorphisms and trauma exposure to predict the development of PTSD (Liberzon *et al.* 2014), which may similarly underlie the relationship between family psychopathology, trauma and offspring diagnoses. Analyses of adoptees have suggested a combination of both biological parental history with adoptive parental experiences lead to increased risk of outcomes such as offspring hospitalization due to suicide attempt (Wilcox *et al.* 2012), highlighting the importance of potential interactions between genetics and familial environment. Further analyses can investigate potential genetic effects, whether through single polymorphisms or more general polygenic risk scores, to evaluate the relationship between childhood traumatic experiences and later psychiatric diagnoses. Similarly, important familial factors which may have an impact on exposure to traumatic experience and may be related to parental psychopathology, such as parental physical or psychiatric impairment, stress, neglect or decreased monitoring of child behavior, can also be investigated using these latent classes.

Psychiatric outcomes associated with class membership underscore that specific early traumatic experiences are associated with distinct consequences, including, but not limited to PTSD. The women in the Sexual Assault class were more likely to experience global impairment in terms of increased suicidal thoughts and behaviors as well as increased incidence of almost all of the examined psychiatric diagnoses, when compared to the women in the low trauma

class. Men in the Violence Exposure class had more circumscribed effects, with increased rates of ASPD and PTSD compared to men in the low trauma class. Results suggest that patterns of childhood traumatic experiences may be associated with specific transdiagnostic vulnerabilities (Teicher & Samson, 2013), which may be related to potential epigenetic changes as well as neurobiological pathways such as reduced hippocampal volume or amygdala reactivity. Further investigation of potential epigenetic or neurobiological differences between the three classes may highlight distinct neurodevelopmental pathways to psychiatric outcomes.

Out of the other negative outcomes included in this model, only STIs and traumatic events in young adulthood were consistently associated with class membership. STIs were closely associated with the Sexual Assault class; it is not known whether these infections were transmitted as a result of the assaults in childhood or in young adulthood or related to other sexual behavior. With the exception of stomach problems, which have been associated with stress and anxiety (Means-Christensen *et al.* 2008), none of the other lifetime health outcomes were associated with class membership. Only non-violent criminal convictions were associated with Violence Exposure class when analyses were stratified by gender, suggesting that the association between childhood trauma and outcomes such as alcohol and drug use disorders or ASPD did not extend to legal repercussions. Class membership was associated with traumatic experiences in young adulthood, particularly for the Sexual Assault class, in which young adult women were more likely to experience sexual assault, physical assault and witness death. These results replicate previous work in this area (Acierno *et al.* 1999) and highlight the importance of early intervention for survivors of child trauma. As data collection ended at age 29 years, it is not known how these traumatic experiences in young adulthood then impacted further experiences in middle adulthood and beyond.

Several characteristics of the study limit the findings. First, traumatic experiences reported in young adulthood may have been subject to reporting biases, particularly by gender. Although we may not have captured all possible trauma events given this methodological approach, research suggests that the false positive disclosures of trauma are unlikely (Hardt & Rutter, 2004). Additionally, the perpetrator of the trauma was not recorded; therefore, it is not known whether the traumatic exposure was instigated by someone in the family or in the larger community. Second, the order of onset among multiple psychiatric outcomes was not evaluated. For example, it is possible that individuals developed PTSD, which put

them at risk for other psychiatric outcomes, such as suicide attempts (Wilcox *et al.* 2009). Third, physical health outcomes and parental psychopathology and substance abuse were not confirmed by clinician or medical record diagnosis. Reported family history of psychiatric diagnoses is limited, but may be more reliable for diagnoses such as depression, schizophrenia or substance abuse (Hardt & Franke, 2007). Fourth, the sample size did not permit further regression models of the impact on gender on psychopathology; there were too few women in the Violence Exposure class and too few men in the Sexual Assault class to investigate possible interactions. Further investigations of the role of gender on both reporting trauma as well as the development of negative psychiatric outcomes after traumatic experiences, is indicated. Fifth, endorsement of many traumatic experiences was quite low, which may have taxed the computation involved in estimating the LCA. Finally, it is possible that the combination of trauma exposure in childhood and young adulthood may have led to adverse psychiatric outcomes by age 30. It is also possible that a traumatic experience occurring after childhood could have been considered a 'worse' experience and more directly led to the psychiatric outcomes. Further analysis using these classes of childhood trauma exposure with the addition of adolescent and young adult trauma exposure is indicated to determine the effect of cumulative trauma.

### Conclusions

Latent classes of childhood traumatic experiences before the age of 13 years were computed and studied using an urban community sample followed from childhood to age 29 years. Assaultive traumatic experiences in childhood, both sexual and non-sexual, were found to predict negative psychiatric outcomes by young adulthood, including re-traumatization. Childhood sexual abuse in our sample predicted more global impairment in women, whereas violence exposure in men predicted more limited outcomes. In our sample, we identified unique developmental pathways by type of childhood trauma exposure to young adult negative psychiatric and behavioral outcomes.

### Supplementary material

For supplementary material accompanying this paper visit <http://dx.doi.org/10.1017/S0033291715001300>.

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### Declaration of Interest

None.

### References

- Acierno R, Resnick H, Kilpatrick DG, Saunders B, Best CL** (1999). Risk factors for rape, physical assault, and posttraumatic stress disorder in women: examination of differential multivariate relationships. *Journal of Anxiety Disorders* **13**, 541–563.
- Anacker C, O'Donnell KJ, Meaney MJ** (2014). Early life adversity and the epigenetic programming of hypothalamic-pituitary-adrenal function. *Dialogues in Clinical Neuroscience* **16**, 321–333.
- Anda RF, Felitti VJ, Bremner JD, Walker JD, Whitfield C, Perry BD, Dube SR, Giles WH** (2006). The enduring effects of abuse and related adverse experiences in childhood. *European Archives of Psychiatry and Clinical Neuroscience* **256**, 174–186.
- Andrews G, Peters L** (1998). The psychometric properties of the Composite International Diagnostic Interview. *Social Psychiatry and Psychiatric Epidemiology* **33**, 80–88.
- Anthony J** (1994). Comparative epidemiology of dependence on tobacco, alcohol, controlled substances, and inhalants: basic findings from the National Comorbidity Survey. *Experimental and Clinical Psychopharmacology* **2**, 244–268.
- APA** (2000). *Diagnostic and Statistical Manual of Mental Disorders*, 4th edn. American Psychiatric Association: Washington, DC.
- APA** (2013). *Diagnostic and Statistical Manual of Mental Disorders*, 5th ed. American Psychiatric Association: Washington, DC.
- Barrish HH, Saunders M, Wolf MW** (1969). Good Behavior Game: effects of individual contingencies for group consequences on disruptive behavior in a classroom. *Journal of Applied Behavior Analysis* **2**, 119–124.
- Block J, Burns R** (1976). Mastery learning. In *Review of Research in Education* (ed. L. Shulman). Peacock: Itasca, IL, pp. 3–49.
- Breslau N, Kessler RC, Chilcoat HD, Schultz LR, Davis GC, Andreski P** (1998a). Trauma and posttraumatic stress disorder in the community: the 1996 Detroit Area Survey of Trauma. *Archives of General Psychiatry* **55**, 626–632.
- Breslau N, Kessler RC, Peterson EL** (1998b). PTSD assessment with a structured interview: reliability and concordance with a standardized clinical interview. *International Journal of Methods in Psychiatric Research* **7**, 121–127.
- CDC** (2015). High school youth risk behavior survey data, 1991–2013. Centers for Disease Control and Prevention (<http://nccd.cdc.gov/youthonline>). Accessed 1 April 2015.
- CDC** (2014). Prevalence of individual adverse childhood experiences. Centers for Disease Control and Prevention

- (<http://www.cdc.gov/violenceprevention/acestudy/prevalence.html>). Accessed 1 April 2015.
- Chaffin M, Kelleher K, Hollenberg J** (1996). Onset of physical abuse and neglect: psychiatric, substance abuse, and social risk factors from prospective community data. *Child Abuse and Neglect* **20**, 191–203.
- Chung H, Breslau N** (2008). The latent structure of post-traumatic stress disorder: tests of invariance by gender and trauma type. *Psychological Medicine* **38**, 563–73.
- Cloitre M, Stolbach BC, Herman JL, van der Kolk B, Pynoos R, Wang J, Petkova E** (2009). A developmental approach to complex PTSD: childhood and adult cumulative trauma as predictors of symptom complexity. *Journal of Traumatic Stress* **22**, 399–408.
- Collins LM, Lanza ST** (2009). *Latent Class and Latent Transition Analysis: With Applications in the Social, Behavioral, and Health Sciences*. Wiley: NY.
- Dong M, Anda RF, Felitti VJ, Dube SR, Williamson DF, Thompson TJ, Loo CM, Giles WH** (2004). The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. *Child Abuse and Neglect* **28**, 771–784.
- Dube SR, Fairweather D, Pearson WS, Felitti VJ, Anda RF, Croft JB** (2009). Cumulative childhood stress and autoimmune diseases in adults. *Psychosomatic Medicine* **71**, 243–250.
- Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Spitz AM, Edwards V, Koss MP, Marks JS** (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine* **14**, 245–258.
- Garner AS, Shonkoff JP** (2012). Early childhood adversity, toxic stress, and the role of the pediatrician: translating developmental science into lifelong health. *Pediatrics* **129**, e224–e231.
- Graham JW** (2009). Missing data analysis: making it work in the real world. *Annual Review of Psychology* **60**, 549–576.
- Grant BF, Dawson DA, Stinson FS, Chou PS, Kay W, Pickering R** (2003). The Alcohol Use Disorder and Associated Disabilities Interview Schedule-IV (AUDADIS-IV): reliability of alcohol consumption, tobacco use, family history of depression and psychiatric diagnostic modules in a general population sample. *Drug and Alcohol Dependence* **71**, 7–16.
- Hardt J, Franke P** (2007). Validity, reliability and objectivity of the family history method in psychiatry: a meta-analysis. *European Psychiatry* **22**, 49–58.
- Hardt J, Rutter M** (2004). Validity of adult retrospective reports of adverse childhood experiences: review of the evidence. *Journal of Child Psychology and Psychiatry* **45**, 260–273.
- Kellam SG, Brown CH, Poduska JM, Ialongo NS, Wang W, Toyinbo P, Petras H, Ford C, Windham A, Wilcox HC** (2008). Effects of a universal classroom behavior management program in first and second grades on young adult behavioral, psychiatric, and social outcomes. *Drug and Alcohol Dependence* **95** (Suppl. 1), S5–S28.
- Kellam SG, Werthamer-Larsson L, Dolan LJ, Brown CH, Mayer LS, Rebok GW, Anthony JC, Laudolff J, Edelsohn G** (1991). Developmental epidemiologically based preventive trials: baseline modeling of early target behaviors and depressive symptoms. *American Journal of Community Psychology* **19**, 563–584.
- Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE** (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry* **62**, 593–602.
- Kessler RC, McGonagle KA, Zhao S, Nelson CB, Hughes M, Eshleman S, Wittchen HU, Kendler KS** (1994). Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. results from the National Comorbidity Survey. *Archives of General Psychiatry* **51**, 8–19.
- Liberzon I, King AP, Ressler KJ, Almlil LM, Zhang P, Ma ST, Cohen GH, Tamburrino MB, Calabrese JR, Galea S** (2014). Interaction of the ADRB2 gene polymorphism with childhood trauma in predicting adult symptoms of posttraumatic stress disorder. *JAMA Psychiatry* **71**, 1174–1182.
- Lo Y, Mendell N, Rubin D** (2001). Testing the number of components in a normal mixture. *Biometrika* **88**, 767–778.
- McLachlan G, Peel D** (2000). *Finite Mixture Models*. Wiley: New York, NY.
- Means-Christensen AJ, Roy-Byrne PP, Sherbourne CD, Craske MG, Stein MB** (2008). Relationships among pain, anxiety, and depression in primary care. *Depression and Anxiety* **25**, 593–600.
- Mehta D, Klengel T, Conneely KN, Smith AK, Altmann A, Pace TW, Rex-Haffner M, Loeschner A, Gonik M, Mercer KB, Bradley B, Muller-Myhok B, Ressler KJ, Binder EB** (2013). Childhood maltreatment is associated with distinct genomic and epigenetic profiles in posttraumatic stress disorder. *Proceedings of the National Academy of Sciences USA* **110**, 8302–8307.
- Muthén LK, Muthén BO** (2013). *Mplus User's Guide, 7th edition*. Muthén & Muthén: CA.
- National Center for Health Statistics** (1994). *Plan and Operation of the Third National Health and Nutrition Examination Survey, 1988–94*. Vital Health Statistics System, Center for Disease Control and Prevention: Alexandria, VA.
- Oggers CL, Jaffee SR** (2013). Routine versus catastrophic influences on the developing child. *Annual Review of Public Health* **34**, 29–48.
- Shonkoff JP, Garner AS** (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics* **129**, e232–e246.
- Storr CL, Or F, Eaton WW, Ialongo N** (2014). Genetic research participation in a young adult community sample. *Journal of Community Genetics* **5**, 363–375.
- Storr CL, Reboussin BA, Anthony JC** (2004). Early childhood misbehavior and the estimated risk of becoming tobacco-dependent. *American Journal of Epidemiology* **160**, 126–130.
- Szyf M** (2011). The early life social environment and DNA methylation: DNA methylation mediating the long-term impact of social environments early in life. *Epigenetics* **6**, 971–978.
- Teicher MH, Samson JA** (2013). Childhood maltreatment and psychopathology: a case for ecophenotypic variants as

- clinically and neurobiologically distinct subtypes. *American Journal of Psychiatry* **170**, 1114–11133.
- Verona E, Sachs-Ericsson N** (2005). The intergenerational transmission of externalizing behaviors in adult participants: the mediating role of childhood abuse. *Journal of Consulting and Clinical Psychology* **73**, 1135–1145.
- Walsh C, MacMillan H, Jamieson E** (2002). The relationship between parental psychiatric disorder and child physical and sexual abuse: findings from the Ontario Health Supplement. *Child Abuse and Neglect* **26**, 11–22.
- Walsh C, MacMillan HL, Jamieson E** (2003). The relationship between parental substance abuse and child maltreatment: findings from the Ontario Health Supplement. *Child Abuse and Neglect* **27**, 1409–1425.
- Wilcox HC, Anthony JC** (2004). The development of suicide ideation and attempts: an epidemiologic study of first graders followed into young adulthood. *Drug and Alcohol Dependence* **76** (Suppl.), S53–S67.
- Wilcox HC, Kuramoto SJ, Brent D, Runeson B** (2012). The interaction of parental history of suicidal behavior and exposure to adoptive parents' psychiatric disorders on adoptee suicide attempt hospitalizations. *American Journal of Psychiatry* **169**, 309–315.
- Wilcox HC, Kellam SG, Brown CH, Poduska JM, Ialongo NS, Wang W, Anthony JC** (2008). The impact of two universal randomized first- and second-grade classroom interventions on young adult suicide ideation and attempts. *Drug and Alcohol Dependence* **95** (Suppl. 1), S60–S73.
- Wilcox HC, Storr CL, Breslau N** (2009). Posttraumatic stress disorder and suicide attempts in a community sample of urban American young adults. *Archives of General Psychiatry* **66**, 305–311.
- Wittchen HU** (1994). Reliability and validity studies of the WHO – Composite International Diagnostic Interview (CIDI): a critical review. *Journal of Psychiatric Research* **28**, 57–84.
- World Health Organization** (1995). *Physical Status: The use and Interpretation of Anthropometry*. World Health Organization: Geneva, Switzerland.
- World Health Organization** (1997). *World Health Organization Composite International Diagnostic Interview (CIDI) Core Version 2.1, Interviewer's Manual*. World Health Organization: Geneva, Switzerland.